FILE 'HOME' ENTERED AT 12:17:59 ON 03 MAR 2003)

FILE 'CAPLUS, BIOSIS, SCISEARCH, MEDLINE, EMBASE' ENTERED AT 12:18:30 ON 03 MAR 2003

L1	16870	S	CREB
L2	0	S	L1 (A) FLUORESCENCE
L3	1039	S	L1 AND FUSION
L4	45	S	L3 AND FLUORESCENCE
L5	28		DUPLICATE REMOVE L4 (17 DUPLICATES REMOVED)

L5 ANSWER 28 OF 28 MEDLINE
AN 94301408 MEDLINE
DN 94301408 PubMed ID: 7913207
TI Nuclear protein CBP is a coactivator for the transcription factor **CREB**.
CM Comment in: Nature. 1994 Jul 21;370(6486):177
AU Kwok R P; Lundblad J R; Chrivia J C; Richards J P; Bachinger H P; Brennan R G; Roberts S G; Green M R; Goodman R H
CS Vollum Institute, Oregon Health Sciences University, Portland 97201.
SO NATURE, (1994 Jul 21) 370 (6486) 223-6.
Journal code: 0410462. ISSN: 0028-0836.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199408
ED Entered STN: 19940818
Last Updated on STN: 19970203
Entered Medline: 19940805
AB The transcription factor **CREB** binds to a DNA element known as the cAMP-regulated enhancer (CRE). **CREB** is activated through phosphorylation by protein kinase A (PKA), but precisely how phosphorylation stimulates **CREB** function is unknown. One model is that phosphorylation may allow the recruitment of coactivators which then interact with basal transcription factors. We have previously identified a nuclear protein of M(r)265K, CBP, that binds specifically to the PKA-phosphorylated form of **CREB**. We have used **fluorescence** anisotropy measurements to define the equilibrium binding parameters of the phosphoCREB:CBP interaction and report here that CBP can activate transcription through a region in its carboxy terminus. The activation domain of CBP interacts with the basal transcription factor TFIIB through a domain that is conserved in the yeast coactivator ADA-1 (ref. 8). Consistent with its role as a coactivator, CBP augments the activity of phosphorylated **CREB** to activate transcription of cAMP-responsive genes.

5 ANSWER 20 OF 28 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 2001:114853 BIOSIS
DN PREV200100114853
TI In vivo visualization of regulation and expression of CRE-mediated transcription during the formation of long-term memory.
AU Kida, S. (1); Iwamoto, T.; Masushige, S.; Silva, A. J.
CS (1) Tokyo University of Agriculture, Setagaya-ku, Tokyo Japan
SO Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-652.4. print.
Meeting Info.: 30th Annual Meeting of the Society of Neuroscience New Orleans, LA, USA November 04-09, 2000 Society for Neuroscience . ISSN: 0190-5295.
DT Conference
LA English
SL English
AB The synthesis of new proteins plays an essential role in LTM as well as long-term potentiation (LTP). Both cAMP and Ca2+ signal transduction pathways are thought to regulate these gene expressions. Members of the **CREB**/ATF families activate cAMP responsive element (CRE)-mediated transcription in response to increases in the intracellular concentration of cAMP and Ca2+. **CREB** is phosphorylated at serine 133 (S133) by PKA and CaMK. Phospho-**CREB** can then interact with transcription coactivator CBP. This molecular interaction is thought to be a crucial step in **CREB**-dependent transcription initiation. Using a traditional KO strategy (**CREB**-alpha/-DELTA), as well as with a inducible system (LBDG521R-CREBS133A), we showed that **CREB** -dependent transcription is required for formation of LTM. However, molecular mechanisms underlying CRE-mediated transcription during the formation of LTM remain unknown. Therefore, we are trying to visualize and monitor CRE-mediated transcription in vivo by using derivatives of the green fluorescent protein (GFP). To visualize the regulation of CRE-mediated transcription, we measured **fluorescence** resonance energy transfer (FRET) using two **fusion** protein transiently expressed in Cos1 cells (with or without PKA): enhanced cyan fluorescent protein (ECFP) fused with CBP (CFP-CBP) and enhanced yellow fluorescent protein (EYFP) fused with **CREB** (YFP-**CREB**). We detected evidence for an interaction between CFP-CBP and YFP-**CREB** that is dependent both on PKA and on **CREB** phosphorylation at S133. To monitor the CRE-mediated transcription activity, we have also derived transgenic mice with CRE-reporter gene. Enhanced green fluorescent protein (EGFP) and beta-galactosidase were used as reporter proteins. We will present the results of these analyses.

5 ANSWER 16 OF 28 CAPLUS COPYRIGHT 2003 ACS
AN 2000:175828 CAPLUS
DN 132:218859
TI A synthetic **fusion** protein for monitoring protein phosphorylation and its use in detecting protein phosphorylation and screening for modulators
IN Hagiwara, Masatoshi; Inouye, Satoshi; Nagai, Yasuo
PA Center for Advanced Science and Technology Incubation, Ltd., Japan
SO PCT Int. Appl., 45 pp.
CODEN: PIXXD2
DT Patent
LA Japanese
IC ICM C07K002-00
ICS C12N015-10; C12Q001-02; G01N033-50; C12Q001-68
CC 7-1 (Enzymes)
Section cross-reference(s): 3
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000014108	A1	20000316	WO 1999-JP4769	19990902
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2341153	AA	20000316	CA 1999-2341153	19990902
AU 9954481	A1	20000327	AU 1999-54481	19990902
EP 1118618	A1	20010725	EP 1999-940632	19990902
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				

PRAI JP 1998-248861 A 19980902
WO 1999-JP4769 W 19990902
AB A synthetic **fusion** protein for monitoring protein phosphorylation, its recombinant expression, a method for detecting protein phosphorylation in vivo, and for screening kinases and compd. promoting or inhibiting phosphorylation are reported. The protein contains a phosphorylation site linked to Aequorea victoria green fluorescent proteins on both sides. Phosphorylation of the protein causes a change in the three-dimensional structure and thus a change in **fluorescence** emission characteristics of the GFPs. Two phosphorylation monitoring proteins by having **CREB** transcription factor phosphorylation site and Kemptide phosphorylation site, resp. between Aequorea victoria RS-GFP and BS-GFP were constructed and expressed in E. coli BL21 cells. While both of these proteins were phosphorylated by A-kinase, only the one with **CREB** phosphorylation site displayed a change in **fluorescence** emission characteristics. COS-7 cells were transformed with **CREB** phosphorylation site protein expression vector and phosphorylation by A-kinase was monitored real time in vivo. The effect of A-kinase specific inhibitor H-89 on phosphorylation was examd. using the system.
ST **CREB** phosphorylation site Aequorea green fluorescent protein; phosphorylation monitoring **fusion** protein screening kinase inhibitor activator

ANSWER 13 OF 28 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

AN 2001:504606 BIOSIS

DN PREV200100504606

TI CCAAT/enhancer binding protein alpha assembles essential cooperating factors in common subnuclear domains.

AU Schaufele, Fred (1); Enwright, John F., III; Wang, Xia; Teoh, Cheryl; Srihari, Roopali; Erickson, Robin; Macdougald, Ormond A.; Day, Richard N.

CS (1) University of California, San Francisco, CA, 94143-0540: freds@metabolic.ucsf.edu USA

SO Molecular Endocrinology, (October, 2001) Vol. 15, No. 10, pp. 1665-1676. print.
ISSN: 0888-8809.

DT Article

LA English

SL English

AB The transcription factor CCAAT/enhancer binding protein alpha (C/EBPalpha) is the DNA binding subunit of a multiprotein complex that regulates the pituitary-specific GH promoter. C/EBPalpha is absent from the GHFT1-5 pituitary progenitor cell line in which ectopic C/EBPalpha expression leads to activation of the otherwise dormant GH promoter. Transcriptional regulatory complexes are commonly envisaged as assembling from components that evenly diffuse throughout the nucleoplasm. We show that C/EBPalpha, expressed in GHFT1-5 cells as a **fusion** with color variants of the green fluorescent protein (GFP), concentrated specifically at peri-centromeric chromosomal domains. Although we found the **CREB** -binding protein (CBP) to activate C/EBPalpha-dependent transcription, CBP was absent from the pericentromeric chromatin. C/EBPalpha expression was accompanied by the translocation of endogenous and ectopically expressed CBP to pericentromeric chromatin. The intranuclear recruitment of CBP required the transcriptional activation domains of C/EBPalpha. C/EBPalpha also caused GFP-tagged TATA binding protein (TBP) to relocate to the Hoechst-stained domains. The altered intranuclear distribution of critical coregulatory factors defines complexes formed upon C/EBPalpha expression. It also identifies an organizational activity, which we label "intranuclear marshaling," that may regulate gene expression by determining the cooperative and antagonistic interactions available at specific nuclear sites.